Relation of Phasic Coronary Flow Velocity Profile to Clinical and Hemodynamic Characteristics of Patients With Aortic Valve Disease

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Background. Our objective was to assess phasic coronary blood flow and velocity characteristics of the proximal portion of the left anterior descending artery and to evaluate their relation to the clinical and hemodynamic manifestations in patients with aortic valve disease.

Methods and Results. We examined 26 patients with chronic aortic regurgitation (AR), 12 patients with predominant aortic stenosis (AS), and 11 control subjects using an intravascular Doppler catheter with spectral analysis. Angiographic assessment of AR identified 10 patients with mild regurgitation and 16 with severe regurgitation. The resting systolic coronary flow velocity-time integral (VTI) was significantly higher and the diastolic VTI was slightly but significantly higher in patients with severe regurgitation than in those with mild regurgitation (11.8±4.2 vs 4.1±1.1 cm, P<.001; 18.5±5.8 vs 13.2±3.2 cm, P<.05) and control subjects (4.0±1.0 cm, P<.001 and 13.3±3.6 cm, P<.05), respectively. Patients with AS had a slightly lower resting systolic VTI (3.8±1.4 cm) and a higher diastolic VTI (14.6±3.7 cm) than control subjects. Resting coronary blood flow was greater in patients with aortic valve disease than in control subjects. There was a significant correlation between the ratio of the resting systolic to diastolic VTI (S/D ratio) and the ratio of the aortic systolic to diastolic pressure (r=.75, P<.001) in patients with AR. The S/D ratio was inversely correlated with left ventricular systolic pressure (r=−.92, P<.001) and positively correlated with the ratio of the aortic systolic to diastolic pressure (r=.68, P<.05) in patients with AS.

Conclusions. Our results indicate that hemodynamic changes related to aortic valve disease contribute to alterations in the resting phasic coronary blood flow and velocity profiles observed in these patients.


KEY WORDS • blood flow • catheters • aorta • regurgitation • stenoses

Recent advances in a catheter-tip Doppler technique for measuring coronary flow velocity have made it possible to assess the functional capacity of coronary arteries to dilate in response to various stimuli in the catheterization laboratory, enhancing our understanding of the coronary circulation in a wide variety of cardiac disease states.1-6 In patients with chronic aortic regurgitation (AR) and those with aortic stenosis (AS), left ventricular (LV) hypertrophy secondary to chronic volume and pressure overload is usually associated with a reduced coronary vascular reserve7-13 and is suggested as a cause of episodes of myocardial ischemia despite normal epicardial coronary arteries. Recently, several investigators, using a Doppler catheter, have reported a predominant systolic component of the coronary flow velocity in patients with chronic AR14,15 and a reduced systolic component with an enhanced diastolic component in patients with AS.14,16 To the best of our knowledge, however, there have been no precise reports on whether the phasic coronary flow velocity profile is related to hemodynamic findings in patients with aortic valve disease.

The present study was designed to analyze phasic coronary flow velocity patterns of the proximal portion of the left anterior descending artery with an intravascular Doppler catheter with a fast Fourier transform (FFT) of the velocity signal to assess the relation of coronary blood flow and velocity characteristics to the clinical and hemodynamic manifestations in patients with aortic valve disease.

Methods

Patients

We examined 26 patients with chronic AR (14 men and 12 women 33 to 68 years old; mean, 52±12 years) and 12 patients with AS (7 men and 5 women 46 to 60 years old; mean, 53±7 years). Of the 12 patients with AS, 8 had pure AS and the remaining 4 exhibited predominant AS combined with mild AR. All patients with aortic valve disease had normal coronary arteries. We also examined 11 patients (7 men and 4 women 35 to 63 years old; mean, 48±12 years) who were referred...
to our hospital for the evaluation of chest pain. They underwent coronary arteriography and left ventriculography and were found to have normal coronary arteries, LV ejection fraction, and wall motion. Systemic blood pressure, chest roentgenograms, ECGs, and M-mode and two-dimensional echocardiograms were also normal. These 11 patients served as the control subjects. All subjects exhibited sinus rhythm at the time of study. Written informed consent was obtained from each subject.

**Cardiac Catheterization**

All cardiovascular medications were withheld for at least 48 hours before cardiac catheterization. Right heart catheterization was performed with a 7F balloon-tipped flow-directed thermocatheter, and left heart catheterization was carried out with a 7F high-fidelity micromanometer (SPC-474A, Millar, Houston, Tex). Mean pulmonary capillary wedge pressure, LV systolic and end-diastolic pressures, and aortic systolic, diastolic, and mean pressures were measured. Mean coronary perfusion pressure was calculated as mean aortic pressure minus mean LV diastolic pressure. Cardiac output was determined by the thermodilution method. LV end-diastolic and end-systolic volumes and ejection fraction were calculated from a single-plane left ventriculogram using the area-length method. AR was estimated by aortography and graded semiquantitatively. Grades I and II were classified as mild AR, and grade III or above was classified as severe AR. The aortic regurgitant fraction was determined by comparing the total stroke volume calculated from the left ventriculogram and the stroke volume obtained by the thermodilution methods immediately before the performance of left ventriculography. The aortic valve area was calculated according to the method of Gorlin and Gorlin, and the aortic valve peak systolic pressure gradient was measured in patients with AS.

**Echocardiography**

Echocardiographic examination and cardiac catheterization were performed on the same day. LV mass was calculated according to the method recommended by the American Society of Echocardiography.

**Coronary Flow Velocity Measurements**

After routine diagnostic cardiac catheterization had been performed, coronary flow velocity was measured in all patients by use of a 3F intravascular pulsed Doppler catheter with an end-mounted Doppler crystal (DC-201, Millar) and a 20-MHz velocimeter (MDV-20, Millar). A pulse repetition frequency was 62.5 kHz. The sample volume was 0.46 mm in depth, and range gate varied from 1 to 10 mm from the catheter tip. The Doppler sample volume was set at 3 to 4 mm beyond the catheter tip. The other features of the Doppler catheter and signal analysis system have been described elsewhere. The components were linked by a custom-designed gain control to a FFT spectrum analyzer (SSH-160A, Toshiba, Tokyo) so that the Doppler shift signals could be analyzed by complex spectral analysis in real time. Signals were recorded on a strip-chart recorder at paper speeds of 50 to 100 mm/s. The Doppler catheter was inserted with a 0.014-in flexible angioplasty guide wire through an 8F guiding catheter. The system, with the guide wire extending beyond the tip of the Doppler catheter, was carefully advanced into the proximal portion of the left anterior descending artery. The catheter position and Doppler range gate were adjusted to obtain an optimal audio signal and a phasic tracing of maximal coronary flow velocity. After the resting coronary flow velocity was measured, maximal hyperemic flow velocity was induced by the intracoronary injection of 10 to 12 mg of papaverine to determine the coronary flow reserve. Coronary arteriography was then repeated to measure coronary arterial diameter.

**Data Analysis**

The Doppler velocity signals were analyzed with the aid of a computer-interfaced graphic analyzer (CARDIO 500, Kontron, Tokyo). Systole was defined as the phase between the top of the R wave on the ECG and the dicrotic notch of the aortic pressure; diastole was defined as the phase between the dicrotic notch and the top of the following R wave. The segment of the coronary artery interrogated by the Doppler catheter was analyzed, and coronary arterial diameter was measured at end diastole by quantitative coronary arteriography using the semiautomatic computer system (CARDIO 500, Kontron). The following variables, expressed as the average of three consecutive cardiac cycles, were obtained: (1) resting systolic (VTLs) and diastolic (VTLd) coronary flow velocity-time integrals, defined as the area under the velocity curve during systole and the area under the curve during diastole; (2) VTLs and VTLd during maximal hyperemia; (3) peak flow velocities of retrograde flows; (4) the area over the retrograde flow velocity curve; (5) systolic plus diastolic velocity-time integrals (VTLs+d) at rest and during maximal hyperemia; (6) coronary vasodilator reserve, defined as the ratio of the estimates of coronary blood flow during maximal hyperemia to the estimates of the flow at rest; (7) estimates of coronary blood flow (Q) were calculated as follows: Q=VTLs+d×cross-sectional area (CSA) of the coronary artery×heart rate; CSA = π×(d/2)², where d represents the coronary arterial diameter; and (7) systolic and diastolic coronary vascular resistances calculated as the ratio of the systolic and diastolic aortic pressures to the coronary flow, respectively.

**Statistical Analysis**

Data are expressed as mean±SD. The differences among groups were analyzed by ANOVA. Correlations between phasic coronary flow velocity and clinical and hemodynamic findings were assessed by linear regression analysis. A probability value of P<.05 was accepted as statistically significant.

**Results**

**Clinical and Hemodynamic Findings**

Of the 26 patients with chronic AR, 10 had mild regurgitation (mean regurgitant fraction, 33±10%; range, 19% to 48%) and 16 showed severe regurgitation (mean regurgitant fraction, 64±3%; range, 59% to 70%) (Table 1). In patients with AS, the aortic valve area ranged from 0.22 to 1.18 cm² (mean, 0.52±0.28 cm²), and the aortic valve peak systolic pressure gradient ranged from 20 to 115 mm Hg (mean, 65±30 mm Hg). Age, heart rate, mean pulmonary capillary wedge pressure, mean aortic pressure, and mean coronary perfusion pressure did not differ significantly among subjects. Angina pectoris was associated with
exertion in 8 (67%) of the 12 patients with AS, whereas anginal pain was related to exertion in only 3 (12%) of the 26 patients with AR.

Coronary Flow Velocity Measurements of Epicardial Artery

Resting VT1s in the epicardial artery was significantly higher and VT1p was slightly but significantly higher in the patients with severe AR than in the patients with mild AR and the control subjects, resulting in a significant increase in the ratio of VT1s to VT1p (S/D ratio) in patients with severe AR. There were no significant differences in these measurements between patients with mild AR and control subjects (Fig 1 and Table 2). None of the patients with AR exhibited diastolic retrograde flow.

Resting VT1s was slightly lower and VT1p was higher in patients with AS than in control subjects, resulting in a significantly lower S/D ratio (Fig 2 and Table 2). Midsystolic retrograde flow was observed at rest in 9 of the 12 patients with AS (Fig 2). The aortic valve pressure gradient in the presence of retrograde flow exceeded 49 mm Hg; without retrograde flow, the gradient was <35 mm Hg. The percentage of the area over the retrograde flow velocity curve occurring during systole ranged from 0 to 5% (mean, 3 ± 2) of the area under the systolic and diastolic forward flow velocity curve.

Resting coronary blood flow was greater in the patients with aortic valve disease than in the control subjects. Both the resting VT1s,D and the CSA were significantly greater in patients with severe AR than in control subjects. The resting CSA was also slightly greater in patients with mild AR and was significantly greater in patients with AS than in control subjects, although there was no significant difference in the resting VT1s,D among these three groups (Table 3). The coronary flow per 100 g LV mass at rest was similar in patients with aortic valve disease and control subjects. However, this parameter was reduced during maximal hyperemia in patients with aortic valve disease compared with control subjects (Table 3). Coronary vasodilator reserve was markedly limited in patients with severe AR and those with AS and slightly reduced in patients with mild AR compared with control subjects (Table 3). The reserve in patients with AS and angina pectoris in effort was less than that in patients with AS without anginal pain (1.63 ± 0.26 vs 2.18 ± 0.08, P < .01), whereas there was no significant difference in the reserve between patients with AR and angina and those with AR and no angina (2.08 ± 0.41 vs 2.25 ± 0.51).

Systolic coronary vascular resistance at rest was slightly decreased in patients with mild AR (P < .05) and greatly reduced in those with severe AR (P < .001), whereas the resistance was slightly increased in patients with AS. Diastolic coronary resistance at rest was significantly lower in patients with severe AR (P < .01) and those with AS (P < .01) and slightly lower in patients with mild AR than in control subjects (Table 3).

Relation Between Coronary Flow Velocity Measurements and Clinical and Hemodynamic Findings and Vasodilator Reserve

In patients with AR, there was a significant correlation between the resting S/D ratio and the ratio of the aortic systolic to diastolic pressure (r = .75, P < .001) (Fig 3). The S/D ratio was positively correlated with the aortic regurgitant fraction (r = .83, P < .001) and inversely correlated with coronary vasodilator reserve (r = −.65, P < .001) (Fig 4). Of the 16 patients with severe AR, 10 patients in whom LV end-diastolic volume index was ≥180 mL/m² had slightly but significantly greater resting S/D ratio compared with 6 patients whose index was <180 mL/m² (0.67 ± 0.11 vs 0.57 ± 0.03, P < .05). In patients with AS, there were significant correlations between the resting S/D ratio and LV systolic pressure (r = −.92, P < .001) and between the S/D ratio and the ratio of the aortic systolic to diastolic pressure (r = .68, P < .05) (Fig 5). The S/D ratio was positively correlated

### Table 1. Clinical and Hemodynamic Findings

<table>
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<tr>
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<th>Control</th>
<th>Mild</th>
<th>Severe</th>
<th>AS</th>
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<tr>
<td>No.</td>
<td>11</td>
<td>10</td>
<td>16</td>
<td>12</td>
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<td>Age (y)</td>
<td>48±12</td>
<td>50±6</td>
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<tr>
<td>HR (bpm)</td>
<td>69±9</td>
<td>71±13</td>
<td>75±16</td>
<td>72±11</td>
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<td>Mean PCWP (mm Hg)</td>
<td>9±3</td>
<td>9±4</td>
<td>13±6</td>
<td>11±3</td>
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<tr>
<td>AoP (mm Hg)</td>
<td>124±15</td>
<td>133±15</td>
<td>160±14±4</td>
<td>121±9</td>
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<tr>
<td>Systolic</td>
<td>65±11</td>
<td>60±10</td>
<td>51±9†</td>
<td>70±12</td>
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<td>Diastolic</td>
<td>83±16</td>
<td>84±11</td>
<td>88±13</td>
<td>86±10</td>
</tr>
<tr>
<td>LVP (mm Hg)</td>
<td>130±12</td>
<td>136±22</td>
<td>153±17§</td>
<td>186±33§</td>
</tr>
<tr>
<td>Mean CPP (mm Hg)</td>
<td>75±13</td>
<td>73±8</td>
<td>72±11</td>
<td>69±12</td>
</tr>
<tr>
<td>LVEF</td>
<td>0.75±0.12</td>
<td>0.73±0.17</td>
<td>0.64±0.13*</td>
<td>0.70±0.13</td>
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<tr>
<td>CI (L · min⁻¹ · m⁻²)</td>
<td>3.50±0.37</td>
<td>3.42±0.66</td>
<td>2.91±0.75*</td>
<td>3.51±0.67</td>
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<tr>
<td>LVM (g)</td>
<td>126±22</td>
<td>166±78</td>
<td>335±97§</td>
<td>238±106†</td>
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</tbody>
</table>

AR, aortic regurgitation; AS, aortic stenosis; HR, heart rate; bpm, beats per minute; PCWP, pulmonary capillary wedge pressure; AoP, aortic pressure; LVP, left ventricular pressure; CPP, coronary perfusion pressure; LVEF, left ventricular ejection fraction; CI, cardiac index; LVM, left ventricular mass.

*P < .05 vs control; †P < .01 vs control; ‡P < .001 vs control; §P < .05 vs mild AR; ||P < .001 vs mild AR.
FIG 1. Representative recordings of phasic coronary flow velocity at rest in a patient with mild aortic regurgitation (panel A) and in a patient with severe aortic regurgitation (panel B). The patient with mild regurgitation (regurgitant fraction of 33%) shows a normal diastolic-predominant coronary velocity pattern, with a normal ratio of systolic to diastolic velocity-time integral (0.32), whereas the patient with severe regurgitation (regurgitant fraction of 66%) shows marked augmentation of the systolic component and lesser enhancement of the diastolic component with an increase in the ratio (0.65). AoP, aortic pressure.

rest and during maximal hyperemia and the other clinical or hemodynamic findings.

Discussion

Phasic Coronary Blood Flow and Velocity Patterns in Aortic Valve Disease

In studies of experimental acute AR\textsuperscript{23-29} that investigated phasic coronary blood flow patterns of epicardial coronary arteries using an electromagnetic flowmeter, the percentage of coronary flow occurring during systole increased in relation to the systolic and diastolic coronary perfusion pressures. Conflicting results have been obtained in dogs with acutely induced AS. One study reported a decreased S/D ratio of the coronary flow,\textsuperscript{30} but another found no significant change in the ratio,\textsuperscript{31} although both revealed an augmented mean coronary flow. In patients with chronic AR in whom coronary flow and velocity measurements were made during open heart surgery, the predominant systolic component of the phasic flow pattern of epicardial coronary arteries was replaced by the diastolic-predominant pattern after surgery.\textsuperscript{15,32} In patients with AS, an improvement in abnormal coronary flow waveforms occurred after the valve replacement.\textsuperscript{16} Matsuo et al\textsuperscript{14} studied phasic coronary flow velocity patterns in conscious patients with aortic valve disease using a Doppler catheter with a zero-crossing detector that was posi-

<table>
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<th>Table 2. Doppler-Derived Indexes of Coronary Flow Velocity</th>
<th>AR</th>
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<tbody>
<tr>
<td></td>
<td>Control</td>
</tr>
<tr>
<td>No.</td>
<td>11</td>
</tr>
<tr>
<td>VTIs (cm)</td>
<td>4.0±1.0</td>
</tr>
<tr>
<td>VTId (cm)</td>
<td>13.3±3.6</td>
</tr>
<tr>
<td>S/D ratio</td>
<td>0.30±0.03</td>
</tr>
</tbody>
</table>

AR, aortic regurgitation; AS, aortic stenosis; VTIs, resting systolic velocity-time integral; VTId, resting diastolic velocity-time integral; S/D ratio, ratio of resting systolic to diastolic velocity-time integrals.

*P<.05 vs control; †P<.001 vs control; ‡P<.05 vs mild AR; §P<.001 vs mild AR.
tioned at the coronary orifice. They found that the resting S/D ratio tended to be positively correlated with the angiographic grade of regurgitation in patients with chronic AR.14

A recently developed pulsed Doppler velocimeter using an FFT method can detect characteristic spectral broadening.33-35 More recently, investigators have confirmed experimentally and clinically that absolute coronary flow velocity and the estimation of precise flow velocity patterns can be measured accurately by the catheter-tip Doppler technique used in conjunction with a spectral analysis system and by using spectral peak flow velocity rather than mean velocity.21,36,37 We therefore used peak flow velocity to calculate VTIs in the present study.

With increasing regurgitation in patients with AR, we found a shift of resting coronary flow and velocity in the epicardial artery from diastole to systole that corresponded to the changes in coronary perfusion pressure. This pattern was more prominent in patients with marked LV dilatation than in those with mild dilatation. Thus, epicardial phasic coronary flow and velocity waveforms are influenced mainly by changes in coronary perfusion pressure in patients with AR. In experimental and clinical studies24,25,27,30 on acute or chronic AR, several investigators have noted a middiastolic backward flow or negative net diastolic coronary flow and a resting S/D ratio >1.0. Although we did not observe such patterns in any patient, they may be attributed to difference in the severity of disease in our patients and those in other studies with more severe regurgitation.

Although patients with AR show systolic-predominant changes in phasic coronary blood flow pattern of epicardial arteries, the phasic nature of intramyocardial blood flow has been reported to be unchanged because an increase in aortic pulse pressure associated with AR augments capacitance effects of the large epicardial arteries, which may distort actual phasic perfusion of the myocardium.39,40 Therefore, it should be recognized that AR produces disparate effects on phasic coronary blood flow pattern in the epicardial arteries and that in the intramyocardial arteries.39,40 Some investigators have proposed the following mechanism by which characteristic alterations in phasic epicardial coronary flow waveform are produced in severe AR25: the low or

![Graph showing relation between the ratio of the resting systolic to diastolic coronary flow velocity-time integral (S/D ratio) and the ratio of aortic systolic to diastolic pressure (AoSP/AoDP ratio) in patients with aortic regurgitation. A significant positive correlation is observed between them.](image-url)
negative coronary blood flow during diastole leaves the epicardial capacitance vessels partially collapsed. Thus, since pulse pressure increases markedly during systole, the epicardial arteries can store more blood before the development of extravascular compressing forces. As the severity of AS increased, the systolic component of coronary blood flow and velocity was decreased and the diastolic component was exaggerated in proportion to the striking increase in the extravascular compressing forces during systole, as suggested by a marked elevation in LV systolic pressure, although a weak correlation was found between the resting S/D ratio and the ratio of the aortic systolic to diastolic pressure. Thus, the extravascular compressing forces may be the major factors that contribute to the altered phasic coronary flow and velocity patterns in patients with AS. Systolic retrograde flow of the epicardial artery was observed in the majority of our patients with moderate-to-severe AS, although it is worth noting that this flow is more prominent in distal and intramyocardial vessels. We found that the reversal of the flow occurred consistently during mid-systole and that the magnitude of the flow, which was closely related to the aortic valve peak pressure gradient, was <5% of the systolic and diastolic forward flows. In contrast, limited coronary vasodilator reserve was accompanied by the clinical observation of angina pectoris with effort. These findings strongly suggest that an impaired coronary vasodilator capacity, not the systolic retrograde flow, plays a particularly important role in the pathogenesis of myocardial ischemia in patients with AS.

Our observation of the increase in the resting coronary blood flow, attributable to the increased VTI over one cardiac cycle and the CSA, in patients with aortic valve disease resembles that reported previously. In addition, resting coronary blood flow per unit mass was similar in patients with aortic valve disease and control subjects, as in several previous reports. This indicates that patients with aortic valve disease are fully compensated at rest and that catheter-based Doppler technique for calculating coronary perfusion is valid. The total myocardial oxygen requirement is augmented as a result of the increased cardiac work load. The combination of an increased oxygen demand and a reduced oxygen supply may be responsible for the development of myocardial ischemia, especially in response to stress, which is manifested by the reduction of the coronary reserve in these patients.

**FIG 4.** Graphs showing relations between the ratio of the resting systolic to diastolic coronary flow velocity-time integral (S/D ratio) and aortic regurgitant fraction (RF) (panel A) and coronary vasodilator reserve (CVR) (panel B) in patients with aortic regurgitation. The ratio is positively correlated with the RF and inversely correlated with the index of CVR.

**FIG 5.** Graphs showing relations between the ratio of the resting systolic to diastolic coronary flow velocity-time integral (S/D ratio) and left ventricular systolic pressure (LVSP) (panel A) and the ratio of aortic systolic to diastolic pressure (AoSP/AoDP ratio) (panel B) in patients with aortic stenosis. The S/D ratio is highly correlated with the LVSP and mildly correlated with the AoSP/AoDP ratio.
Coronary Flow Velocity Patterns in Aortic Valve Disease

**Clinical Implications**

In all patients with chronic AR that we studied, the resting S/D ratio was highly correlated with the aortic regurgitant fraction and moderately correlated with the index of coronary vasodilator reserve. In patients with AS, there were significant correlations between the S/D ratio and the aortic valve area, the aortic valve peak systolic pressure gradient, and the index of the coronary reserve. Thus, the present study of the phasic coronary velocity profile in the epicardial vessels provides clinically useful information on disease severity and coronary vasodilator capacity in patients with aortic valve disease.

**Potential Limitations**

There are several possible limitations to the present study. First, the Doppler catheter itself alters the coronary flow velocity profile across the vessels in the sample volume that is placed near the catheter tip. The catheter underestimates flow velocity in this region. Marked changes in flow volume and pulsatility may further alter the relation between the instantaneous spectral peak velocity and mean velocity. These effects may be exaggerated at the branching and angulation points and in tortuous segments of vessels. Although these may limit the usefulness and reliability of the quantitative flow calculations in our study, we carefully avoided the major branches and the angulations. To minimize the interference effect and thus measure the actual phasic flow more accurately, it would be desirable to use the Doppler-equipped guide wire system. A second consideration is related to the dependence of coronary flow velocity on hemodynamic changes in patients with normal LV function. Some investigators have observed an increase in mean resting coronary flow velocity without a change in mean hyperemic velocity, leading to a reduction in the coronary flow reserve, in conjunction with increases in heart rate and LV preload. Heart rate and pulmonary capillary wedge pressure did not differ significantly among patient groups in our study. Finally, it is possible that the correlations between the coronary flow velocity measurements and hemodynamic indexes were somewhat scattered in patients with AS because of the presence of concomitant mild regurgitation.

**Conclusions**

We analyzed the phasic coronary flow velocity patterns of the proximal portion of the left anterior descending artery using an intravascular Doppler catheter with an FFT analysis system to assess coronary blood flow and velocity characteristics and their relation to the clinical and hemodynamic findings in patients with aortic valve disease. With increasing regurgitation in patients with chronic AR, resting coronary flow and velocity shifted from diastole to systole, corresponding to changes in coronary perfusion pressure. In patients with AS, as the severity of disease increased, the systolic component of the coronary flow and velocity decreased and the diastolic component increased in proportion to the striking increase in LV systolic pressure. Thus, hemodynamic changes related to aortic valve disease consistently altered phasic coronary flow and velocity profiles in these patients.

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